Case Report

Sudden collapse after minor head injury in an elderly man; association with cardiocerebral decompensation and fat embolism syndrome

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Abstract

An 86-year-old man suffering from right hemiparesis and dementia fell from a stretcher and sustained laceration and bruising to his right eyebrow. He was brought to an emergency room, and his wounds were treated. Shortly after discharge, his respiratory and mental status dramatically declined. Despite supportive care, he died about three hours after re–admission. Autopsy revealed a minor laceration to the eyebrow with minor intracranial injuries, mild cardiomegaly (370 g) with right atrial dilatation, pericardial effusion (170 ml) and marked pulmonary edema. Microscopically, fatty droplets were observed in the lung capillaries, the glomeruli and the intracerebral vessels. Although the extent of the original injuries seemed insufficient to cause systemic fat embolism syndrome (FES), the patient's decreased cardiac reserves and stress associated with this event likely contributed to his death consistent with the physiochemical model of FES pathogenesis.

1. Introduction

The physical and physiologic reserves of elderly people are reduced compared to younger people and, otherwise routine medical conditions can lead to profound morbidity and mortality in the elderly. This is especially true of patients in long-term medical or nursing-care facilities, and forensic pathologists are often asked to differentiate between the relative contributions of acute versus chronic conditions following death in these patients. We present the case of an elderly, bedridden patient who suffered a rapid deterioration and death following a relatively "minor" injury.

2. Case report

An 86-year-old male accidentally fell from a stretcher while taking an assisted bathing in a nursing home, and experienced a laceration at his right eyebrow. He previously suffered from right hemiparesis, motor aphasia and vascular dementia secondary to old cerebral hemorrhages and he had been confined to his bed for fourteen years. After the accident, the patient was pale and dyspneic, and he was transported to a general hospital. Cranial CT scan showed no apparent new cerebral lesions, and chest X-ray revealed congestive heart failure. Oxygen saturation was 82–83% breathing room air, and he was given inhaled oxygen during wound suturing. The patients' respiratory status was improved, and the attending physician allowed his transport back to the nursing home. Soon after getting into the car, however, the

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patient once again became pale, and his mental status declined precipitously. The patient was emergently admitted to the hospital. Upon admission, the patient was wheezing and in atrial fibrillation with a heart rate of approximately 150 beats per minute. Despite supportive care, the patient remained unconscious and died about three hours after admission.

3. Autopsy findings

The decedent weighed 44.0 kg. He had a kyphosis and an accurate height could not be recorded. Autopsy showed 2.3 cm and 2.5 cm T-shaped lacerations on the right eyebrow arch with circumjacent 6×3 cm subcutaneous hemorrhages, and right black eye with 2.5 cm and 1.5 cm linear fractures on the right orbital part of the frontal bone. No other fractures were found on the calvarium. The brain, 1150 g in weight, had large old encephalomalacia in the left hemisphere, subarachnoid hemorrhages within narrow ranges on the right frontal and occipital lobes, small contusion on the right frontal lobe and a small amount of subdural hematoma on the interhemispheric fissure. The lateral ventricles were dilated and two hundred milliliter of cerebrospinal fluid was collected. There were no herniations or secondary hemorrhages in the brain. Separation of the osteophyte occurred between the bodies of the C5–C6 cervical vertebrae, but the vertebral disc and cervical cord remained intact.

Some new abrasions and bruises were present on the trunk and extremities, but no severe injuries, including rib fractures, were present in the thoraco-abdominal cavities. The heart, 370 g in weight, had mild focal fibrosis in the posterolateral wall, right atrial dilation, and atherosclerosis of anterior interventricular branch and right coronary artery with no more than 50% of stenosis. We could collect 170 ml of clear yellowish pericardial effusion. The lungs, left 270 g and right 480 g in weight, were moderately congested and markedly edematous.

4. Histopathology

Interstitial fibroses were scattered across the myocardium, but no apparent recent infarcts were observed. The epicardium was mildly infiltrated by lymphocytes.

Using the combination of frozen sectioning and Sudan III staining to randomly-sampled specimens from both lungs, a plethora of rounded fat globules were found to be disseminated throughout the mid-level vessels and the capillaries in every microscopic field (Fig. 1). There were no inflammatory lesions in the lungs. The similar fat globules were also scattered in the glomeruli (Fig. 2a) and intra-cerebral vessels (Fig. 2b).

5. Discussion

The deceased had a mild cerebral contusion, subarachnoid hemorrhages and subdural hematoma. Additionally, a relatively large volume of cerebrospinal fluid was collected, suggesting impaired absorption secondary to a previous cerebral infarction and postoperative adhesion followed by chronic elevated intracranial pressure. However, no findings suggested lethal intracranial hypertension, such as apparent herniations and secondary brainstem hemorrhages. The patient could have compensated for chronically elevated intracranial pressure, and age-related atrophy and existing cerebral atrophy could have contributed to the reduced brain volume. Thus, we could not determine whether these injuries contributed to the patient's death.

The decedent was an 86-year-old man with mild cardiac hypertrophy with interstitial fibrosis, suggesting decreased cardiopulmonary reserves. Moreover, the presence of a pericardial effusion and atrial fibrillation, likely disrupted diastolic filling and exacerbated the deceased's cardiopulmonary failure.[1] Nonetheless, it seemed to be insufficient to account for his death by cardiac failure only, because we found no evidence of recent myocardial damage or valvular heart disease that is consistent with sudden deteriora-
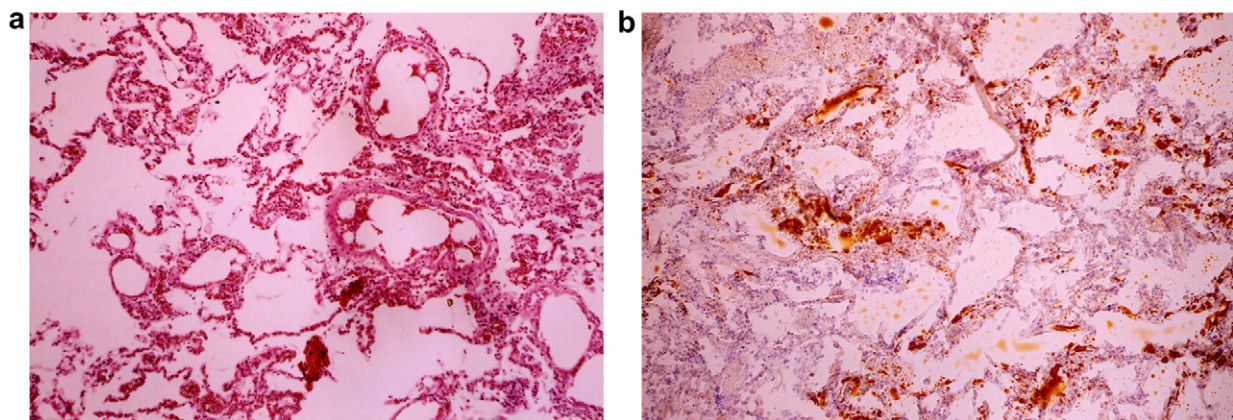

Fig. 1. Pulmonary capillaries with numerous fat globules. (a) formalin-fixed and paraffin-embedded section, Hematoxylin and Eosin staining; (b) formalin-fixed frozen section, Sudan III staining.
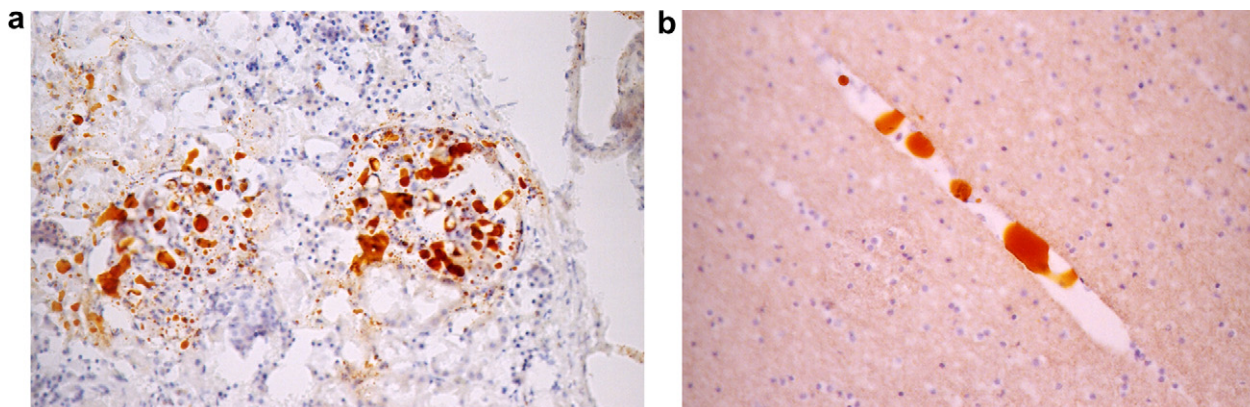
Fig. 2. (a) Glomeruli trapping fat globules (formalin-fixed frozen section, Sudan III staining); (b) fat emboli in the cerebral vessels (formalin-fixed frozen section, Sudan III staining).

tion of his condition. Thus, we had initially tried to put his death down to the combination of cerebral and cardiac dysfunction.

However, microscopic multiple round-shaped fat globules were detected in lung vessels. According to the histological grading of Falzi et al.[2] this finding was classified into distinct fat embolism. Fat globules in the lung alone may develop during cardiovascular resuscitation,[3] but we could not detect any signs of cardiac massage including rib fractures in this case. In contrast, the presence of systemic fat globules in glomeruli and intracerebral capillaries suggests a vital reaction,[4] and the deceased most likely suffered from antemortem systemic fat embolism syndrome (FES). In patients afflicted with systemic FES, autopsy findings are common including marked pulmonary edema and subconjunctival and intracerebral petechiae.[5] However, Hiss et al. argues that petechiae do not have to be present.[6]

FES occurs in approximately 60% of traumatic deaths.[6] FES is regarded as a major complication of fracture,[3,7–10] but can be associated with soft tissue injuries without fractures,[3,4,10,11] burns and other diseases (fatty liver, infection, diabetes mellitus, acute pancreatitis, osteomyelitis and shock).[5,7–12] In this case, no apparent major fractures were detected and soft tissue injuries were almost completely limited to the small area around the site of injury. These injuries would not seem sufficient to cause systemic FES at first glance. However, there is controversy about the correlation between the severity of FES and the degree of injury. Hiss et al. reviewed 32 cases of FES and concluded that there was no correlation between FES outcome and the severity of the inciting injury.[6] Hamood et al. reported a case of a patient who fell from a modest height and who presented FES with severe hypoxemia ($PaO_2 = 32$ mmHg) despite the absence of obvious internal and external trauma.[10]

Two mechanisms have been proposed to account for the development and pathogenesis of fat emboli.[5,7–10] The mechanical theory predicts that destruction of fat cells by tissue injury produces fat droplets that then occlude the vascular lumen impairing gas exchange in the lung capillaries. In contrast, the physiochemical theory predicts that tissue injury generates bioactive substances such as cathecholamines, which cause intravascular mobilization and agglutination of fat, followed by intrapulmonary fatty acid production and tissue damage akin to acute respiratory distress syndrome. These two theories are not mutually exclusive, however, and could both account for some of the observed pathology in FES. In the present case, the occurrence and progression of FES could have arisen from both the head injury but also the pain and emotional stress subsequent to physical injury.

Nichols et al. presented a case of FES complicated by severe systemic soft tissue injury,[7] and they suggested that soft tissue hemorrhages followed by hypovolemia contributed to the patient's death. Hiss et al. also suggest that forensic pathologists should not determine FES as the cause of death without completely excluding other causes while considering the extent of fat embolization, the causative injury, and preexisting conditions. Likewise, Ohya and Bunai advocate limiting conclusions about the relationship between the death and the number or the shape of emboli.[8] Thus, FES could be one of several factors contributing to death in an individual patient. In the present case, there was evidence of systemic FES, so we assumed that the physical and emotional stress had caused not only cardiocerebral decompensation but also systemic FES, resulting in a vicious circle which led to his demise.

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